# Letter to the Editor

## Cutis marmorata and cerebral arterial gas embolism

Dr Kemper and colleagues reported that, when air was injected into the cerebral circulation of pigs, they developed a rash that looked very similar to *cutis marmorata* of cutaneous decompression illness (DCI) and to *livido reticularis*.[1] They postulated that cutaneous DCI in divers may be centrally mediated as a result of cerebral gas embolism.

It would be helpful if Kemper et al. described the distribution of the rash in their pigs. In divers, cutaneous DCI is generally confined to parts of the body with significant amounts of subcutaneous fat, such as the trunk and thighs, and the rash often crosses the midline.

Colleagues and I have reported that cutaneous DCI is commonly associated with significant right-to-left shunts and particularly persistent foramen ovale (PFO).[2] We postulated that the manifestations of shunt-related DCI, whether neurological or cutaneous, are in large part determined by peripheral amplification of embolic bubbles in those tissues that are most supersaturated with dissolved nitrogen (or other inert gas) at the time that emboli arrive. Hence we postulated that cutaneous DCI is the result of amplification of gas emboli that invade cutaneous capillaries.

Dr Kemper has kindly sent me a number of the publications from his department on which their report of this skin rash in pigs is based. The aim of their experiments was to produce significant brain injury by means of cerebral air embolism. Their pigs had no tissues supersaturated with inert gas. They were ventilated with a $F_iO_2$ of 0.4 and anaesthetised with ketamine and midazolam. They were also given pancuronium and atropine, before air was injected into their cerebral circulation. If their findings in pigs and the resulting hypothesis were applicable to man, it would mean that one could get cutaneous DCI without decompression: one would only need cerebral gas embolism.

During contrast echocardiography, I have produced arterial gas embolism in many hundreds of patients with right-to-left shunts and it is certain that some bubbles went into their cerebral circulations, but I have never seen and no patient has reported getting a rash. Nor am I aware of any reports of gas embolism causing a rash like cutaneous DCI without there being tissue supersaturation following some form of decompression.

Kemper and colleagues injected between 0.25 and 1 $ml \cdot kg^{-1}$ body weight of air into the ascending pharyngeal artery (roughly equivalent to human internal carotid artery) of pigs weighing 30–40kg. That immediately produced significant elevation of blood pressure and heart rate suggesting a 'sympathetic surge'. This is similar to the haemodynamic effects that can occur with subarachnoid haemorrhage and some other catastrophic brain injuries. That effect may have been potentiated by pre-treatment with atropine. There was also a considerable increase in intracranial pressure and major adverse effects on cerebral metabolism. Some pigs died quickly and the survivors were killed at the end of the experiment. I suspect that no pig would have survived the experiments without major neurological injury if they had not been killed.

Most people with cutaneous DCI have no detectable neurological manifestations at the time that they have a rash. In those that do have neurological manifestations, it is rarely catastrophic.

The increases in heart rate and blood pressure reported in the pigs are similar to the effects of a phaeochromocytoma, which can cause *livido reticularis* in man.[3,4] Therefore, I wonder whether an alternative explanation for these observations might be that the cerebral injury in the pigs was so massive that the sympathetic surge was comparable to the effects of catecholamine release from a phaeochromocytoma and caused a rash similar to that seen in patients with a phaeochromocytoma.

**References**


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*Peter T Wilmshurst, Consultant Cardiologist, Royal Stoke University Hospital, Stoke-on-Trent, UK*

***E-mail:*** *<peter.wilmshurst@tiscali.co.uk>*



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